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ACUTE SPLENIC MILIARY TUBERCULOSIS.[1]

By D. D. Stewart, M.D.,
of Philadelphia.

That the tubercular process, whether in an acute miliary or more chronic form, may have its only outward expression in the spleen, and remain there entirely limited for a greater or less period, is a fact little recognized, yet one which has been emphasized by several recorded cases.

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[1] Read at the Sixteenth Annual Meeting of the Association of American Physicians, held at Washington, D. C., April 30 to May 2, 1901.

As a chronic affection presumed primary tubercular splenomegaly is less infrequent than the acute miliary form. Bender,[1] who has recently published an exhaustive paper on the subject of tuberculosis of the spleen, alludes to some seven cases which he regards as instances of the former. In these Bender thought that the tubercular affection of the spleen was in so far limited to this viscus that involvement of other organs or parts gave no clinical manifestation. This, however, is an error concerning at least one of the cases which Bender quotes, in which splenectomy was performed—that of Bland Sutton. In this case Bland Sutton informs me the primary lesion was in the lung, and was of the nature of a " quiescent tuberculous cavity."

In Marriott's case[2] Bender is cognizant that the source of infection was perhaps a chronic vulvar ulcer which had been excised. Marriott curiously had reported this case as one of *acute* tuberculosis of the spleen. It is said there was no clinical indication of tuberculosis elsewhere, even at the end of two years, when a splenectomy was done, and eight months subsequently the patient was in good health.

It is not the purpose of this paper to discuss chronic tuberculous splenomegaly, but to draw especial attention to an acute form of miliary splenic tuberculosis, the occurrence of which, though extremely rare, seems undeniable, and which, because of its rarity, has been doubtless occasionally overlooked. But a solitary case has been recorded—Scharold's[3]—and it is most interesting. It is that of a convict, who, apparently previously in good health and employed in out-of-door labor, was suddenly seized with symptoms suggesting an acute overwhelming general infection. There were high fever, prostration of strength, headache, cough, epistaxis, and diarrhœa, retention of urine, delirium, and general cyanosis. The spleen was greatly enlarged. Death occurred on the seventeenth day. A necropsy showed a miliary tuberculosis limited to the much enlarged spleen. In the writer's case, although he cannot demonstrate that the infectious process was in onset or in early course limited to the spleen, yet he believes it is fair to assume this in view of the interesting symptomatology and because the instance of Scharold's shows that such a limited miliary tuberculous process may occur and actually run its course without other involvement.

The writer's case was one in which more than ordinary interest was taken, both on account of its unique character and because of an acquaintanceship with certain of the patient's relations, one of whom, a physician, under whose observation she had previously been, watched the progress of the ailment with him.

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[1] Gazette des Hôpitaux, March 31 and April 7, 1900.

[2] Lancet, November 23, 1895.

[3] Aerztliches Intelligenz-Blatt, August 7, 1883, No. 32.

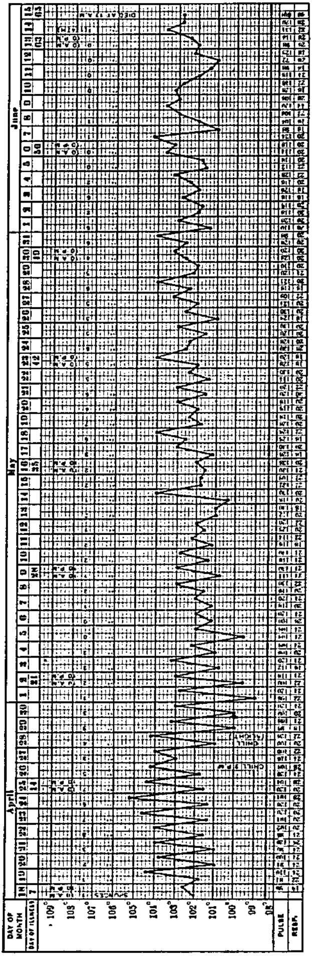
The patient was Emma W., aged twenty-nine years; by occupation a nurse; single; of German parentage and American birth; the fifth of ten children, all living save one killed in a railroad accident; brothers and sisters in good health; parents living; father a dyspeptic, but otherwise well; mother in robust health. The family history otherwise offered nothing noteworthy; no tubercular antecedents.

Previous History. She had had as a child scarlet fever, measles, and pneumonia. Had had ague seventeen years before, but had had no indications of this since. She was of slender build, but quite robust, and had had no illness to incapacitate her for years. About four weeks before admission to the hospital, after having just left a trying and long-continued case of typhoid fever, she had had a slight grippal attack lasting a few days. She was not confined to bed. Dr. Pottberg, her relative, had seen and prescribed for her. One week later, three weeks before admission to the Episcopal Hospital, when not recuperated from the grippe, ill-judged notions of humanity prompted her to take charge of a case of tuberculosis. The patient, living in her vicinity, had just lost his nurse; he was very ill and required constant attention. The consumptive, in indigent circumstances, had apartments which were badly heated and damp, and the nurse's nourishment was of poor quality. She ate irregularly, poorly, and lost sleep. She continued in charge for two weeks. In the middle of this second week she, herself, became rather acutely ill and had chills, with a rise in temperature, was debilitated, and had pain and aching in the dorsal region. She still continued in attendance for a few days despite these symptoms, until a successor could be procured, and then went to her lodgings. There she remained about a week, abed, during which time fever and debility continued. It is stated the temperature then ran between 101° and 103°, and she was presumed by her medical attendant to be developing typhoid fever. She was placed under my care at the Episcopal Hospital on about the tenth day of illness (April 18, 1900), and died the sixty-eighth day, fifty-eight days after admission.

An examination on admission elicited: Mind active, though expression dull, with tendency to somnolence. But neither the dull expression nor somnolent tendency was manifest after a few days spent in hospital. Had had slight chill every day or so since the ailment began.

Abdomen. No pain or tenderness or distention of the bowels. Spleen markedly enlarged, reaching above to sixth interspace, and inferiorly is readily palpable below the costal margin. No typhoid spots. Liver outline normal.

Chest. Heart sounds clear, though enfeebled; no murmurs. A searching examination of the lungs showed total absence of any indication of disease here. This repeated carefully daily gave similarly negative results until late in the progress of the case. The tongue was clean and of healthy appearance. This continued a feature until late in the disease. There were total loss of appetite, constipation, no nose bleed. Temperature, afternoon of admission, 102.6°. On the following day it was 101.4° in the morning, and 104.4° in the evening. It was subsequently taken every third hour throughout the entire course of the case.


DAY OF MONTH
DAY OF ILLNESS
April
May
June
109°
108°
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
PULSE
RESP.
CHILL
DIED AT 12 A.M.

The accompanying (abbreviated) chart shows the daily morning and evening temperature, and that the fever was a continuous one, with morning remissions and evening exacerbations. The average range was between 101° and 104°. The temperature only once reached 99° (in the morning), and but on one occasion (in the evening) 105.2°. The pulse was usually accelerated. The lowest noted was 84, and the highest 140. It showed wide and irregular variations. The number of respirations per minute were commonly in direct ratio to the body temperature.

Widal reaction and examination for plasmodia (Ghriskey) negative. (These examinations were made subsequently on alternate days for two weeks, with similar negative results.) Leucocyte count on admission, 5600; urine, faint albumin trace; microscopically (centrifuged specimen), but one hyaline cast noted; otherwise negative. In arriving at a diagnosis the following ailments were considered: typhoid fever, æstivo-autumnal malarial fever, and acute miliary tuberculosis. The last was almost immediately suspected, and within a few days decided upon, but solely by exclusion, as the general features of the case were unlike those of other cases of acute miliary tuberculosis I had encountered. Here there certainly was as yet no lung involvement, and no signs of abdominal implication other than the presence of the enlarged palpable spleen. The marked splenic enlargement, indeed, was the only notable physical sign other than the persistently high temperature and debility. There were for many days no other noteworthy clinical symptoms, apart from occasional abdominal pain referable to the splenic region, and a tendency to cyanosis and continued blueness of the extremities when cold sponging was resorted to to reduce temperature. The patient's mind was clear and active, and she constantly reproached us for not ridding her of the fever, which she regarded as the only symptom of illness, that she might return to her home. Without other developments the patient's condition underwent little change for the greater part of the fifty-eight days. The lungs were carefully auscultated daily, with negative results, until about two weeks before death.

On May 14th (approximately the thirty-sixth day of the disease) two small glands were palpable in the left supraclavicular fossa. This was the first evident indication of dissemination of the tuberculous process. An ophthalmoscopic examination on this date (Dr. Van Pelt) showed congestion of retinæ of moderate degree limited to each nasal side above the disk, more marked in O. S.

Toward the end of May marked debility was present as a natural result of the continued fever. She took but little nourishment, although free from gastro-intestinal symptoms. At this time fine râles were evident now and then in both lungs. These, with vesiculo-bronchial breathing, were first noted posteriorly, her continued dorsal decubitus favoring stasis in the posterior surfaces of the lungs. On June 1st she had several spells of nausea and vomiting, and pain was complained of in the abdomen. Vomiting now recurred almost daily until death. She became delirious at night and had headache by day. It was noted on June 5th that the general body surface was markedly hyperæsthetic; jerking and twitching of the limbs had occurred the evening before, and she frequently now gave a sharp outcry. She had been slightly deaf for several days, and was now markedly so. On June 12th the bedside notes record: "Deafness less apparent; responds more readily when questioned, though commonly delirious. Apparently is free from unprovoked pain, but remonstrates when the abdomen is palpated. Frequent twitching of upper extremities, and these movements are accompanied by a harsh cephalic cry. Kernig's sign is absent. Tache cerebrale marked. Abdomen much distended. Tongue for first time dry and brown in color, sordes appearing. Pulse intermittent. Yesterday bright red circular area, about one inch in diameter, appeared on the back of the left hand. It had a distinct white border, and was covered with vesicles. The whole hand was swollen and somewhat red. To-day vesicles have united into one very large one, and similar spots have appeared on thumb and fingers. Cultures from the vesicles show staphylococci." On June 15th the notes state: Patient rigid for the past two days. Kernig's sign still absent. Drooping of right eyelid noted. Emaciation now becoming extreme. Area on hand has formed superficial slough. The patient's outcry is less shrill, and jerking of the limbs less marked. She is more or less comatose, constantly moans, and no longer responds rationally to questions. Marked weakness is apparent. Pulse continues intermittent. Patient died cyanosed on the morning of June 15th.

Clinical pathologist's (Dr. Ghriskey) report: No indications of kidney involvement until late in the case. Albumin and casts absent in the early period of the disease. Numerous blood inspections negative, save for a leucocytosis first noted June 12th. Several examinations of the erythrocytes were made, the last May 10th: 3,800,000. Hæmoglobin, estimated on several occasions early in the disease, 65 to 75 per cent. Leucocytes frequently estimated; counts ran between 5000 and 6000 until on June 12th, 14,800 (terminal infection). Malarial parasites, frequently examined for early in the case, always absent. During the first week the blood was also drawn from the spleen for this examination. Unfortunately tubercle bacilli were not examined for in this specimen. Cultures from this (for typhoid) were made; result negative. In the middle period of the disease 4 c.c. of blood from a vein was diluted, centrifuged, and examined for tubercle bacilli; result negative. Several sputum examinations were made the last of May and early in June and no tubercle bacilli found. Diazo reaction examination on several occasions was negative at first, but positive on May 7th. Cultures from vesicles on hand and from meninges of brain showed staphylococci. Other cultures at post-mortem negative.

Report of the necropsy and gross and microscopic examination of specimens by the Episcopal Hospital pathologist, Dr. W. E. Robertson, June 15, 1900: The body is that of a slender young woman. Rigor mortis absent. A large bulla noted on dorsum of left hand.

Thoracic Cavity. Lungs and heart taken out together. Heart presented nothing abnormal. Lungs the seat of diffused, very small miliary tubercles. No consolidation. Lungs crepitated throughout. Moderate pleural adhesion on both sides. Several medium-sized lymph glands in left supraclavicular fossa, enlarged and caseated, and resting upon subclavian artery.

Abdomen. Slight amount of fluid and some injection of intestines. No tubercles on mesentery or peritoneum. Intestines and stomach were examined throughout. No typhoidal or tubercular lesions. A few shallow ulcers or erosions in the ascending colon. Liver slightly fatty but firm, and presented fairly numerous minute tubercles through it.

No gallstones. Duct patulous. Kidney capsules stripped with ease. Section showed cloudy swelling, apparent slight interstitial change. Few small miliary tubercles evident near capsule.

The most noteworthy feature of the examination was the spleen. This was about four times the normal size. It was studded on its peritoneal coat with elevated yellow tubercles the size of a large pea, and surrounded by an inflammatory zone. "On section the whole organ was found to be a mass of tubercles, varying in size from a pin-point to 4 mm. in diameter. These, to about the extent of one-half of the spleen, had advanced to complete caseation, converting this part of the gland into a confluent tubercular mass, in which, while the outline of some of the tubercles was still apparent, in the greater part there was merely evident a most marked cheesy necrosis. Such a condition was found nowhere outside the spleen."

Pancreas normal. Bladder distended. Wall thick-looking, and vessels in it turgescent. Urine blood-tinged. Wide-spread submucous extravasation of blood. "Blood could be neither wiped nor washed off. There was nothing to explain this high grade of hyperæmia, which resembled that met with in some cases of paraplegia, or where the circulation through the inferior cava is interfered with. Such a condition is sometimes seen in hemorrhagic diseases, such as scurvy, and it is possible that in this case the intensity of the infection may have induced it as it did the blood extravasations in the kidney and spleen."

Brain. Dura bulged on removing skullcap. Large amount of fluid escaped when it was cut. It was quite adherent over region of Pacchionian bodies. In this region, too, a slight amount of puriform material was found, and also in a few other places scattered irregularly over the cortex, these latter in the subarachnoid space apparently. Adhesions in fissures at base. Miliary tubercles along small vessels in fissure of Sylvius. Cord not removed.

Histological examination of liver, kidneys, and spleen.

Liver. Parenchymatous degeneration. Slight increase of perilobular connective tissue, and a tendency to multiplication of bile capillaries. Numerous microscopic miliary tubercles, the large majority being in an early stage, only an occasional one presenting some cheesy necrosis. There was no confluence of tubercles.

Kidney. Slight diffuse parenchymatous degeneration. Numerous microscopic miliary tubercles, for the most part early, though there were a few which were decidedly degenerated in their centres, and here and there some coalescence had taken place, so that the mass was visible to the naked eye, though very small.

Spleen presented the most pronounced tuberculous condition far more than any other organ in the body. In addition to countless miliary tubercles in a comparatively early stage, these being far more numerous than found in the other organs, were confluent tubercles, many of them small, others very large, and all in an advanced stage of necrosis, about one-half of the organ being appropriated by this conglomerate tubercle mass. Both kidneys and spleen were much congested and extravasations of blood were common, especially in the spleen.

There is an interesting similarity between the case of the writer and that of Scharold before mentioned. Like Scharold's, my patient became acutely ill, but evidently having greater resisting power, the manifestations of general infection were much less overwhelming, and the duration of the ailment considerably more extended. Scharold's case died on the seventeenth day, mine on the sixty-eighth. As in Scharold's case, enlargement of the spleen was noticeable early. This, with other acute manifestations, had naturally caused the attendant prior to admission to the hospital to regard the ailment as typhoid fever, and the patient was admitted to the hospital with this diagnosis. Unlike Scharold's case,[1] symptoms and physical signs suggestive of pulmonary involvement were totally absent, and their continued absence, with a similar dearth of indications of localization of the tuberculous process elsewhere than in the spleen, was regarded as most singular. As detailed in the history, other than the splenic enlargement and the continuous and rather high temperature, there was a curious absence of symptoms through the greater part of the course of the case. The splenic enlargement reached its maximum rather early, and showed no increase after the third week of illness. The spleen was not notably sensitive to pressure until late in the disease. Unlike Scharold's case (Scharold gives only the macroscopic appearances of the presumed uninvolved organs) the tubercular process had generalized when death occurred. Miliary tubercles were then evident in the brain, lungs, liver, and kidneys. Those in the lungs, though disseminated, were recent and minute, and there were no indications of consolidation. There were also but few tubercles manifest macroscopically in the liver and kidneys, and none existed in the peritoneum or in the gastro-intestinal tract, uterine appendages, or bladder. The process was so extensive and advanced in the spleen, and so extraordinarily out of proportion to that encountered elsewhere, that Dr. Robertson, though unaware of the history, at once remarked the case was evidently one of primary miliary tuberculosis of this organ, and this, I believe, we must so regard it.

Infection in this case apparently arose through the consumptive the patient was nursing. It must, of course, have occurred early in her contact with him, as symptoms appeared acutely about the middle of the second week. She had gone to the case for humanitarian reasons, and, contrary to the advice of her friends, a week after the onset of a mild attack of supposed grippe. Though this had not confined her to the house, she stated she still felt weak. Immediately prior to this she had left a convalescent typhoid patient, in which case she had been the sole nurse, and had continued with it for two months. She was then ill prepared to undertake the arduous duties which devolved upon her in nursing the consumptive. As before remarked, this patient's rooms were damp and cold, and the nurse was constantly in attendance, lost much sleep, and was poorly fed.

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[1] I was then unaware of the case of Scharold. My attention was drawn to it subsequently by Dr. W. E. Robertson.

NOTE UPON A CASE OF CARDIA DUPLEX IN A TURKEY.

By Allen J. Smith, M.D.,
of Galveston, Texas.

(*From the Pathological Laboratory, University of Texas, No. 5, 1901.*)

The specimen upon which this note is based came into the writer's hands in so unfortunately marred a state that many of its interesting features could only be surmised, and for this reason it has seemed advisable to record, without any extended discussion (which under the circumstances could have led to little at best), the occurrence of the anomaly and to briefly describe the condition as far as definitely shown in the specimen.

Several months ago two of the students of the second-year class in medicine in this institution—Messrs. Ferguson and Reifel—brought the writer two hearts which they stated had on the previous day been taken from a turkey in its preparation for the day's meal. The housekeeper in removing the viscera first dragged out the smaller of the two organs, not having recognized its double, and remarked its small size; and in a moment, feeling the larger organ, dragged it forth in turn. Realizing that the condition was a very unusual one, she kept the two specimens, and on the return of the students to their rooms the same evening she brought the hearts to them as a curiosity. In the meantime the turkey had been cooked and eaten. As far as could be learned, however, there was noted nothing in the appearance of the fowl suggestive of any abnormality or disease, and it was said to have been a female turkey of ordinary size.

On examining the specimens, both with the pericardial sac gone and the vessels at the base of each heart torn away (the veins almost entirely torn out of the auricular walls, the arteries persisting for a short distance beyond the cardiac walls), it was evident from the existence of a flattened surface on each where the two organs had been in apposition, and from the correspondence of auricular tears, that the two were originally continuous, one right auricle serving for both and uniting the two organs at the base, as indicated in the diagrams. The larger organ weighed 13.5 grammes, the smaller 4 grammes. The measurements of the larger were as follows: circumference at base, 8 centimetres; length of left ventricle (outside measurement), 4.5 centimetres; thickness of the wall of the left ventricle, 0.8 centimetre; thickness of the wall of the right ventricle, 0.2 centimetre. The measurements of the small organ were as follows: circumference of base, 5 centimetres; external length of left ventricle, 3 centimetres; thickness of wall of left ventricle, 0.4 centimetre; thickness of wall of right ventricle, 0.15 centimetre. The volume and weight proportions are thus seen to be nearly uniform, except that the wall of the left ventricle of the larger heart is proportionately thicker than that of the smaller, and, on the contrary, the wall of the right ventricle of the smaller is proportionately thicker than that of the larger organ.

In the accompanying diagrams the hearts have been drawn as if still united, the first and second figures representing fairly the external appearance of the specimen from ventral and dorsal aspects, the third and fourth figures being merely diagrammatic representations of the interior arrangements of each heart separately. In Fig. 1, a ventral view of the two hearts, the various parts are indicated by letters as follows: *R.V.*, right ventricle of major heart; *A.R.V.*, accessory right ventricle (or right ventricle of minor heart); *L.V.*, left ventricle of major heart; *A.L.V.*, accessory left ventricle; *S.*, septum; *R.A.* and *A.R.A.*, the fused right auricles of the two organs; *L.A.*, left auricle (of major heart only); *P.*, pulmonary artery of major heart; *A.P.A.*, accessory pulmonary artery; *A.*, aorta with traces of three branches; *A.P.*, accessory aorta with two branches. Fig. 2 represents the dorsal view of the same. The letters correspond, and in addition *P.V.* indicates a pulmonary vein entering the left auricle, and *V.C.* one of the venæ cavæ entering the right auricle. The auricular wall was so badly torn that the writer could not be certain of any of the veins except this

FIG. 1.

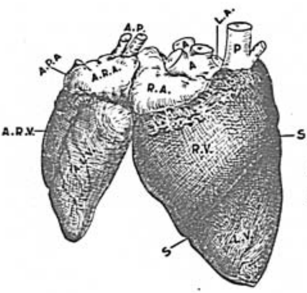


FIG. 2

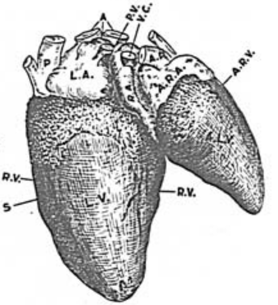
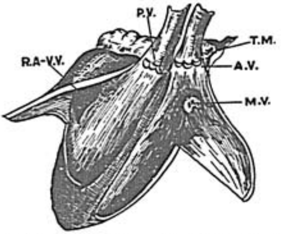


FIG. 1.—Ventral view. *L. V.* Left ventricle. *S.* Interventricular septum. *R. V.* Right ventricle. *A. L. V.* Left ventricle of small organ (accessory left ventricle). *A. R. V.* Accessory right ventricle. *R. A.* and *A. R. A.* Fused right auricles of both organs. *L. A.* Left auricle of large organ. *A.* Aorta from large heart. *A. P.* Aorta from small heart. *P.* Pulmonary artery of large heart. *A. P. A.* Pulmonary artery from small heart.

FIG. 2.—Dorsal view. Letters as above. *P. V.* Pulmonary vein entering left auricle. *V. C.* Vena cava entering fused right auricles.

one pulmonary vein entering the left auricle in its upper portion back of the aorta and close to the interauricular septum; otherwise this part of the wall was apparently without any entering vessels. An irregular hole in the wall of the fused right auricle at *V.C.* (Fig. 2) and another

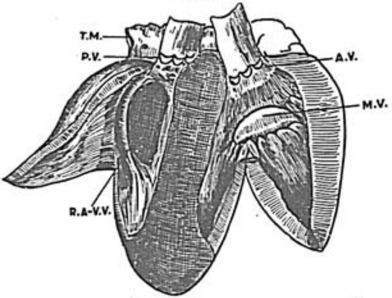
FIG. 3.



Schematic drawing of small heart laid open. *A.V.* Aortic valve. *P. V.* Pulmonary valve. *M. V.* Mitral valve. *R. A-V. V.* Right auriculo-ventricular valve. *T. M.* Torn margin of separated right auricle.

lying slightly ventral to it were accepted tentatively as the points of entrance of the venæ cavæ, and probably of the other veins entering the right auricle. Fig. 3 represents the small heart laid open. The letters indicate respectively: *A.V.*, aortic valve; *M.V.*, mitral valve;

FIG. 4.



Schematic drawing of large heart laid open. Letters as in Fig. 3.

P.V., pulmonary valve; *R.A-V.V.*, right auriculo-ventricular valve; *T.M.*, torn margin of fused right auricle rent in removal of the specimen. Fig. 4 represents a similar view of the interior of the larger organ, and the lettering corresponds with that of the preceding figure.

It is evident that the essential anomaly, aside from the undoubted vascular irregularities lost, was due to a fusion of the two right auricles of otherwise separate double hearts in one individual, with absence of a left auricle in the smaller of the two organs. The course of the blood stream, then, must have been from the systemic veins into the united right auricles; thence through the three openings, easily determined upon exposure of the cardiac interiors, into the two ventricles of the small organ and right ventricle of the larger heart (*R.A-V.V.* and *M.V.* of Fig. 3, and *R.A-V.V.* of Fig. 4). The blood from the two right ventricles probably went to the lung; but it is quite possible that some of the venous blood passing through *A.V.* (Fig. 3) may have been driven from the left ventricle of the small heart into the arterial system. Returning from the lung, the blood probably found its way regularly through the pulmonary vein into the left auricle, and thence, passing through the mitral valve into the left ventricle of the large organ and out through the aortic opening, into the general arterial system. A curious arrangement is to be seen in the construction of the right auriculo-ventricular valve in each organ (*R.A-V.V.*). Instead of the usual three fibrous sheets met in the structure of the tricuspid valve, in each of these hearts such cusps are replaced by a comparatively thick, fleshy fold of the wall of the ventricle, attached above to the auriculo-ventricular septum or close to it, and closely adapting itself against the interventricular septum when closing the opening. In the smaller heart, as represented in Fig. 3, a small fibromuscular band or sheet attached to the auriculo-ventricular and interventricular septa serves to some slight extent as an opposing sheet to this muscular valve. A proportionately smaller, less distinct structure of the same kind is seen as well in the larger organ. In each heart the inner surface of the right ventricle was peculiarly void of the usual pectinate muscles and papillary muscles, and was quite smooth. In the absence of other data the writer prefers to forego discussion and comparison with the occasional similar findings of other observers;[1] but believes the extreme rarity of the anomaly and its functional importance make it worthy of at least this passing notice.

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[1] Gould and Pyle, in their work upon anomalies and medical curiosities, make the following statement: "Duplication of the heart, notwithstanding the number of cases reported, has been admitted with the greatest reserve by Goeffroy-St. Hilaire, and by a number of authors. Among the celebrated anatomists who describe duplex heart are Littre, Meckel, Collomb, Panum, Behr, Paullini, Rhodius, Winslow, and Zacutus Lusitanus. The 'Ephemerides' cites an instance of triple heart, and Johnston has seen a triple heart in a goose."